Finally, DHFS spent $48,500 to monitor potential links between CWD and Creutzfeldt-Jakob disease, which is a fatal neurological disease that afflicts humans. Efforts included a review of death certificates for individuals who were judged to have died from neurological diseases. They were funded to protect human health and provide information on the potential transmission of disease-causing agents from deer to humans.

Future Considerations

A number of questions pertaining to the State's CWD efforts will need to be considered in the future. A primary question is whether DNR has taken the correct approach in its efforts to combat CWD. As noted, the disease was identified in Colorado and other western states before it was found in Wisconsin. However, the size of Wisconsin's deer herd and the potential effects of CWD on deer hunting in the state led Wisconsin to become a national leader in efforts to combat the disease.

Questions have been raised about DNR's policy of eradication, and recently DNR officials have begun using the term "herd reduction" rather than eradication to describe the agency's efforts to depopulate deer within the eradication and intensive harvest zones. They note that eradication refers to their efforts to eliminate the disease and not to kill all of the deer within the eradication or intensive harvest zones, which may not be possible.

However, the animal health and disease research experts with whom we spoke believe that elimination of the disease may still be possible, even if all deer within these zones cannot be killed. They believe it may be premature for the State to move to a system of disease containment rather than eradication. Some disease experts believe that a policy of aggressive herd reduction could lead to a situation in which the concentration of deer within infected areas is reduced to the point that infected deer eventually die without passing the disease on to uninfected animals. However, it is not possible to determine the likelihood of success in the State's efforts to eradicate CWD because too many variables exist at the present time, including prevalence of the disease, its ease of transmission, and the extent to which prions remain in the soil and general environment as disease causing agents.

Even if eradication of the disease does not turn out to be a viable strategy, many believe that DNR's current efforts will prevent or limit the spread of CWD, because depopulation reduces the concentration of deer within areas known to have infected animals, thereby limiting opportunities to transmit the disease. DNR's efforts will need to be supported by DATCP, which will be expected to play an active role in ensuring that infection is not introduced through the importation of deer into areas of the state where the disease does not currently exist.

Second, in order to have any chance of being successful, DNR's strategy of depopulating the deer herd in infected areas must be maintained over time. Mild winters and deer births could rapidly increase the size of the herd within the eradication and intensive harvest zones unless efforts are made to ensure that herd reduction activities are maintained. To that end, DNR announced in September 2003 that it will establish a pilot program, known as the CWD Control Reward Program, under which hunters who kill a CWD infected deer within the eradication or

intensive harvest zones will be paid $200 rewards. The rewards will be funded with $200,000 from the Fish and Wildlife Account of the Conservation Fund and a $50,000 contribution from an anonymous donor. If a deer is harvested on private land, the landowner will also receive a $200 reward. Any funds remaining after the rewards have been paid will be distributed through random drawings in $20 increments to all hunters registering deer from the two zones.

The reward program will be administered by Whitetails Unlimited, Inc., which will oversee the fund, coordinate payments, and conduct the random drawings. Because a private organization will be responsible for the expenditure of public funds, efforts will need to be made to ensure that all expenditures are made in compliance with applicable rules and regulations.

Third, state officials will need to closely monitor the cooperative arrangement of DNR and DATCP relating to the regulation of deer, in order to ensure that obligations and responsibilities are fulfilled and regulatory activities are proceeding smoothly. As noted, DATCP assumed responsibility for the majority of regulatory functions associated with farm-raised deer in January 2003. However, DNR has maintained responsibility for regulating fences that enclose white-tailed deer. In March 2003, the two agencies entered into a formal cooperative agreement through a memorandum of understanding that specifies how they will cooperate in promoting the interests of the State of Wisconsin and its citizens in protecting deer and elk populations, including both free-ranging and farm-raised animals. The agreement is specific and should afford a framework for inter-agency cooperation.

Fourth, attention will need to be given to ensuring that the tissue digester is used to its full advantage. Accurate estimates of the cost to dispose of deer carcasses through tissue digestion are not currently available because all costs, including sludge disposal and labor, are not known at the present time. However, it is anticipated that the cost to dispose of carcasses through tissue digestion will be lower than incineration. Provided that digestion costs are lower than incineration costs, as expected, DNR officials will need to work closely with the Diagnostic Laboratory to ensure that the full capacity of the digester is used to greatest effect in limiting the State's total carcass disposal costs.

Fifth, agency officials will need to closely monitor CWD-related expenditures to ensure that limited funds are used most effectively to address program needs. Some agency officials believe that current resources are inadequate to meet existing demands. For example, DATCP officials believe they need an additional $253,000 to depopulate farm-raised deer on 13 farms located either within the eradication zone or within two miles of the zone. Because of the location of the farms, DATCP officials believe the only practical way to deal with animals on these farms is through depopulation.

DATCP officials also believe the additional funds are needed to gain the compliance of farm owners with future depopulation orders. Without a guarantee of state reimbursement for the animals to be killed, officials contend DATCP will face lengthy and costly court challenges. DATCP is currently involved in five court cases with farm owners protesting DATCP's existing quarantine or depopulation orders. At this time, it is uncertain whether DATCP will pursue a s. 13.10 request through the Joint Committee on Finance to seek additional funding, or wait

until it has been able to better quantify the issues associated with farms located in or near the eradication zone.

Finally, continued attention will need to be given to the potential spread of CWD to humans and livestock. The research conducted to date has found no evidence of transmission to humans and unlikely transmission to cattle and other livestock. However, state agencies will need to monitor relevant research to ensure that public health and agricultural interests are adequately protected. In June 2003, the University of Wisconsin-Madison announced that that it will receive three grants from the Department of Defense, totaling $5.2 million, to conduct research into the molecular and environmental aspects of CWD, including its potential transmission to other species, as well as soil contamination leading to the spread of the disease. Funds from these grants will be distributed to the university over a five-year period.

****

WISCONSIN LEGISLATIVE COUNCIL

Terry C. Anderson, Director
Laura D. Rose, Deputy Director

TO: SENATOR NEAL KEDZIE

FROM: John Stolzenberg, Chief of Research Services

RE: Assembly and Senate Versions of 2003 Assembly Bill 519, Relating to Feeding of Deer and Elk

DATE: October 22, 2003

INTRODUCTION

This memorandum responds to your questions relating to the version of 2003 Assembly Bill 519 passed by the Assembly and the version of this bill recommended by the Senate Committee on Environment and Natural Resources on the following:

- The effect of the enactment of either of these versions of Assembly Bill 519 on the emergency rules adopted by the Department of Natural Resources (DNR) on September 8, 2003 pertaining to the regulation of deer feeding and baiting (Emergency Order WM-37-03(E)).

- Whether either of these versions of Assembly Bill 519, if enacted, require further rule-making by the DNR to be implemented.

Assembly Bill 519 relates to the feeding of deer and elk. The Assembly-passed version of Assembly Bill 519 (the "Assembly version") is set forth in Assembly Substitute Amendment 1, as amended by Assembly Amendment 1 to Assembly Substitute Amendment 1. The version of Assembly Bill 519 recommended by the Senate Committee on Environment and Natural Resources (the "Senate version") is the Assembly version as amended by Senate Amendment 1, as amended by Senate Amendment 1 to Senate Amendment 1.

Both the Assembly version and Senate version of Assembly Bill 519 are described in the attached Legislative Council Amendment Memo on Assembly Bill 519, dated October 22, 2003.

Your questions arise out of the differences between the regulations set forth in the DNR's emergency rules and the provisions in the Assembly and Senate versions of Assembly Bill 519. The

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emergency rules were promulgated under broad grants of authority to the DNR, and both versions of Assembly Bill 519 narrow this authority.

Prior to addressing your questions, the memorandum provides background information on general principles relevant to this memorandum of statutory interpretation and the relation between an authorizing statute and a rule implementing or interpreting the authorizing statute. The responses to your questions follow and are divided between the provisions in the Assembly and Senate versions relating to the geographic area in which the DNR may promulgate rules prohibiting the feeding of deer for viewing or hunting purposes and the provisions in these versions relating to the allowable locations of feeding sites within these areas and the amounts of feed allowed at a feeding site.

STATUTORY INTERPRETATION AND RULE-MAKING

General principles of statutory interpretation and related rule-making implementing a statute relevant to this memorandum include:

- An agency must have statutory authority to promulgate a rule. This authority may be general or specific.

- A rule interpreting or implementing a statute is not necessary if the statute does not require rule-making for implementation and is clear on its face and can be implemented without further interpretation. (However, under this circumstance, agencies often promulgate the statutory requirement in a rule so that the agency's rules on the subject contain a complete set of relevant regulations.)

- If there is a conflict between a requirement in a statute and a rule interpreting or implementing a statute, the statutory requirement prevails over the requirement in the rule.

- If the statutory authority for a particular rule is amended after an agency has promulgated the rule under the original statutory authority, any part of the existing rule in conflict with the new authority no longer has effect since there is no authority for that part.

DISCUSSION

Geographic Area of Feeding Bans

Assembly Version

The provisions in the Assembly version of Assembly Bill 519 that relate to the geographic area of feeding bans are set forth in proposed s. 29.336 (2). While there is some ambiguity over the interpretation of s. 29.336 (2) (intro.), which is discussed below under the Senate version, this ambiguity does not need to be addressed in reviewing the Assembly version of these provisions. This conclusion follows from the observation that the criteria for designating which geographic areas the feeding bans apply for both viewing and hunting purposes are the same in the DNR's emergency rule and the Assembly version. (Both use the same criteria based on entire counties.)

As such, the DNR's current emergency rules satisfy any rule-making authorization or requirement in s. 29.336 (2), and this portion of the Assembly version of Assembly Bill 519 would serve to ratify or support the emergency rules and require no changes in the rules.

Senate Version

The provisions in the Senate version of Assembly Bill 519 that relate to the geographic area of feeding bans are set forth in proposed s. 29.336 (2), as created by the Assembly version and amended by the Senate amendments to the bill. The language in s. 29.36 (2) (intro.) in the Senate version that states "the department may promulgate rules that prohibit feeding deer for hunting or viewing purposes in any of the following [areas specified in pars. (a) to (c)]" is ambiguous.

This ambiguity arises because normally such grants of rule-making authority are prospective and granted before the administering agency has promulgated any rules. In this instance, the DNR has already promulgated rules that prohibit feeding deer for hunting or viewing purposes and some of the areas in which feeding deer is banned under the rules do not match the areas specified in the Senate version. In particular, the DNR's emergency rules specify that the feeding of deer may not occur in a county in which a positive test for chronic wasting disease or bovine tuberculosis has been confirmed in any captive or free-roaming domestic or wild animal in the county after December 31, 1997 or that has any portion within a 10-mile radius of such a positive test. The Senate version replaces these criteria with the criterion that the feeding ban applies in any city, village, or town in which the municipality or portion of the municipality is within a 15-mile radius of such a positive test. The Senate version does not change the criterion in the rules for a feeding ban in a county that contains a chronic wasting disease control zone designated by DNR rule.

One reading of the rule-making authority in s. 29.336 (2) in the Senate version is that, since this authority is discretionary (it uses the phrase "may promulgate"), the DNR may choose to not exercise this authority and instead rely upon other authority to support its emergency rules in their present form. This reading appears less defensible than either of the following two readings given below because it fails to recognize the linking by the Senate version of the DNR's general authority to promulgate rules regulating recreational and supplemental feeding of wild animals for purposes other than hunting in s. 29.335 to the specific restrictions created by the Senate version in s. 29.336. This linkage is created by the amendment in the Senate version to s. 29.335 which states that the rule-making duty in s. 29.335 is "subject to s. 29.336."

A second reading of the rule-making authority in s. 29.336 (2) in the Senate version is that, if the DNR chooses to designate areas in which feeding deer would be prohibited, then these areas must be identified by rule and the rule must conform to the criteria given in the Senate version. If the DNR chooses to not designate areas in which the feeding of deer would be prohibited, and thus allow feeding statewide under the restrictions in other parts of the Senate version (s. 29.336 (3) and (4)), then it could have no rules designating banned feeding areas.

For the first option in the second reading to be implemented, rule-making by the DNR would be required. In particular, the DNR would have to amend its current emergency rules so that the criteria relating to a positive test for chronic wasting disease or bovine tuberculosis based upon proximity to counties would conform to the corresponding criterion in the Senate version based upon proximity to municipalities. Based upon the general principles of statutory interpretation given above, until the rules

were amended the only criterion with legal effect would be the criterion in the emergency rules that are also in the Senate version. This criterion establishes a feeding ban in any county that contains a chronic wasting disease control zone designated by DNR rule.

For the second option in the second reading to be implemented, in which the DNR would designate no area in the state for a deer feeding ban, the DNR would have to remove from its existing emergency rules the feeding ban in any county that contains a chronic wasting disease control zone designated by DNR rule.

A third reading of the rule-making authority in s. 29.336 (2) in the Senate version is that, though the rule-making authorized in s. 29.336 (2) is a discretionary power, given the recent history of the state regulation of feeding deer, including the enactment of 2001 Wisconsin Act 108 and the objection by various standing committees in the Legislature to the DNR's original feeding rules, the legislative intent is that s. 29.336 (2) establishes a duty of the DNR to promulgate feeding bans by rule in the designated areas.

As indicated, implementation of this reading requires rule-making by the DNR. To the extent that the criteria identifying areas where there is a feeding ban in the existing emergency rules do not conform to the criterion in the Senate version, as indicated in the general principles of statutory interpretation given above, upon enactment of the Senate version those criteria in the rules would have no effect. The criterion in the Senate version that differs would not take effect until the DNR promulgated a rule containing the criterion. The other criterion in the rules (that applies the feeding ban to any county that contains a chronic wasting disease control zone designated by DNR rule) that conforms to the same criterion in the Senate version would continue to apply after enactment of the Senate version of Assembly Bill 519.

Allowable Location and Size of Feeding Sites

Assembly Version

The provisions in the Assembly version of Assembly Bill 519 that relate to the allowable locations and size of feeding sites in areas where feeding is not banned are set forth in proposed s. 29.336 (3) and (4). The Assembly version does not specify that these provisions have to be implemented by rule. To the extent that the DNR determines that they do not require further interpretation to be implemented, each provision can stand on its own.

Where a provision in the Assembly version conflicts with a corresponding provision in the DNR's emergency rules, then under the general principles of statutory interpretation provided at the beginning of this memorandum, the statutory provision would supersede the provision in the rules. An example of this conflict is the allowable amount of feed at a feeding site used for hunting purposes. The rules authorizes up to 10 gallons of bait in s. NR 10.07 (2m) 2., Wis. Adm. Code. The Assembly version authorizes no more than two gallons of material at a feeding site in s. 29.336 (4) (a). As noted above, the DNR could choose to amend its rules to remove such conflicts and create a complete and current set of applicable feeding regulations, though from a legal point of view this reconciliation is not necessary.

Where a provision in the Assembly version contains a requirement that is not in the rules, the requirement will apply upon enactment of the bill, irrespective of whether it is in the rules. An example of this situation is the requirement in s. 29.336 (3) (b) that a feeding site for viewing purposes must be not less than 100 yards from a roadway having a posted speed limit of 45 miles per hour or more. Again, the DNR could choose to amend its rules to include the new requirement and create a complete and current set of applicable feeding regulations, though from a legal point of view this addition is not necessary.

Where a provision in the DNR's emergency rules contains requirement that is not in the Assembly version, the provision in the rules continues to apply. An example of this situation is the requirement in s. NR 10.07 (2m) 3. that a person may not place or hunt over bait within 50 yards of any trail, road, or camp site used by the public.

Senate Version

Since the provisions in the Senate version that relate to the allowable location and size of feeding sites in areas where feeding is not banned have the same form and content as the Assembly version, with the additional restriction that deer feed may not contain animal parts or byproducts, the discussion of the Assembly version of these provisions also applies to the Senate version of these provisions.

If you have any additional questions on Assembly Bill 519, please feel free to direct them to me at the Legislative Council staff offices.

JES:tlu:ksm;rv

Attachment

WISCONSIN LEGISLATIVE COUNCIL
AMENDMENT MEMO

2003 Assembly Bill 519	Senate Amendment 1, as Amended by Senate Amendment 1, to Senate Amendment 1
Memo published: October 22, 2003	*Contact*: John Stolzenberg, Chief of Research Services (266-2988) and Mark C. Patronsky, Senior Staff Attorney (266-9280)

Assembly Bill 519

Assembly Bill 519 authorizes any person to feed deer or elk for "viewing purposes" in any area that the DNR has not designated as a chronic wasting disease eradication zone, management zone, or intensive harvest zone. The bill imposes restrictions on the allowable location of feeding and amount of feed.

Assembly Bill 519 authorizes any person to feed deer or elk for hunting purposes north of U.S. Highway 54 during any open season and in any area that the DNR has not designated as a chronic wasting disease eradication zone, management zone, or intensive harvest zone. The bill imposes restrictions on the allowable location of baiting and the amount of bait.

Assembly-Passed Version of Assembly Bill 519

The Assembly passed Assembly Bill 519, as amended by Assembly Substitute Amendment 1, as amended by Assembly Amendment 1 to Assembly Substitute Amendment 1 (hereafter, the "Assembly version").

The Assembly version applies only to deer and not to deer and elk.

The Assembly version authorizes the DNR to promulgate rules that prohibit feeding deer for hunting or viewing purposes in a county which meets any of the following conditions:

- The county or a portion of the county is in a chronic wasting disease control zone designated by DNR rule.

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- A positive test for chronic wasting disease or bovine tuberculosis has been confirmed in any captive or free-roaming domestic or wild animal in that county after December 31, 1997.

- The county or a portion of the county is within a 10-mile radius of the known location of a captive or free-roaming domestic or wild animal that has been tested and confirmed positive for chronic wasting disease or bovine tuberculosis after December 31, 1997.

The method for authorizing the DNR to prohibit the feeding of deer, on a county-by-county basis, as described above, is the same method used by DNR to ban feeding wild animals in emergency rules promulgated on September 8, 2003.

The Assembly version authorizes any person to feed deer for viewing purposes in areas other than the counties in which the DNR prohibits feeding of wild animals, subject to all of the following conditions:

- The feeding site must be within 50 yards of an owner-occupied residence or a person's business, if the business is generally open to the public.

- The feeding site may not be less than 100 yards from the traveled portion of a highway that has a posted speed limit of 45 miles per hour or more.

- No more than two gallons of material may be present at the feeding site at any time.

The Assembly version repeals the current July 1, 2004 sunset of DNR authority to regulate the feeding of wild animals for purposes other than hunting.

The Assembly version authorizes any person to feed deer for hunting purposes in areas other than the counties in which the DNR prohibits feeding of wild animals, subject to all of the following conditions:

- No more than two gallons of material may be present at a feeding site at any time.

- No feeding site may be closer than 100 yards of another feeding site.

- The person does not place more than two gallons of feed in an area of 40 acres or less.

Senate Amendment 1 and Senate Amendment 1 to Senate Amendment 1

Senate Amendment 1 modifies the geographic areas in which the DNR may prohibit by rule feeding deer for hunting or viewing purposes. The amendment replaces the criteria in the Assembly version, described above, that authorizes the DNR to ban the feeding of deer by rule in a county in which a positive test for chronic wasting disease or bovine tuberculosis has been confirmed in any captive or free-roaming domestic or wild animal in the county after December 31, 1997 or that has any portion within a 10-mile radius of such a positive test. In particular, the amendment authorizes the feeding ban in any city, village, or town in which the municipality or a portion of the municipality is within a 10-mile radius of such a positive test. Senate Amendment 1 does not change the criterion in the

Assembly version for an area to be subject to a feeding ban based on a county that contains a chronic wasting disease control zone designated by DNR rule.

Senate Amendment 1 to Senate Amendment 1 modifies this criterion in Senate Amendment 1 to apply the feeding ban rules to any municipality that has a portion of the municipality within a 15-mile radius of such a positive test rather than a 10-mile radius.

Senate Amendment 1 also establishes that an additional condition for feeding deer for viewing or hunting purposes in areas where this feeding is not banned is that the material used to feed deer may not contain any animal part or animal byproduct.

Legislative History

Assembly Substitute Amendment 1 was offered by Representatives Gunderson, M. Williams, and Sherman on September 16, 2003. Assembly Amendment 1 to Assembly Substitute Amendment 1 was offered by Representative M. Williams on September 16, 2003.

Assembly Amendment 1 to Assembly Substitute Amendment 1 was recommended by the Assembly Committee on Natural Resources on a vote of Ayes, 10; Noes, 1. Assembly Substitute Amendment 1 was recommended by the Assembly Committee on Natural Resources on a vote of Ayes, 11; Noes, 0. Assembly Bill 519, as amended, was recommended by the Assembly Committee on Natural Resources on a vote of Ayes, 10; Noes, 1.

The Assembly adopted Assembly Amendment 1 to Assembly Substitute Amendment 1 and Assembly Substitute Amendment 1, as amended, on separate voice votes on September 23, 2003. The Assembly passed Assembly Bill 519, as amended, on a vote of Ayes, 84; Noes, 15, on September 23, 2003.

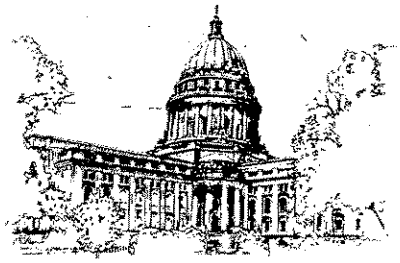
On October 21, 2003, the Senate Committee on Environment and Natural Resources introduced and recommended adoption of Senate Amendment 1 to Senate Amendment 1 and recommended adoption of Senate Amendment 1 on separate votes of Ayes, 3; Noes, 2. The Senate Committee on Environment and Natural Resources recommended concurrence in Assembly Bill 519, as amended, on October 21, 2003 on a vote of Ayes, 3; Noes, 2.

JES:MCP:wu;ksm

WAYNE W. WOOD
State Representative
44th Assembly District

COMMITTEE ASSIGNMENTS
Member:
Rules
Criminal Justice

Ranking Minority Member:
State Affairs
Ways and Means



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April 30, 2003

APR 30

State Representative Glenn Grothman
Room 15 North – State Capitol
Madison, WI 53702

Dear Representative Grothman: Glenn

When the Assembly was debating the bill to give the DNR the authority to "regulate" deer feeding (not ban), I had an amendment prepared that would not have allowed the DNR to ban small amounts of feeding. I did not introduce the amendment because of the attached letter from Tom Hauge indicating they did not intend to seek a total ban.

I thought you would be interested in this information.

Sincerely,

Wayne

WAYNE W. WOOD
State Representative
44th Assembly District

Attachment

5/14/03
Please copy for
members of the
Committee
Thank you
Senator Grothman

DNR
5-15-02
(CWD - Rules)

Wayne

Regarding feeding, we are concerned with the large amounts of food being placed on the landscape to congregate large numbers of deer. We will need common sense rules. It is my intent to recommend to the NRB that we do not ban the placement of a small amount food close to a dwelling so the occupant can view deer.

Tom Hauge

Motion on feeding wild animals

MOVED, that the Senate Committee on Environment and Natural Resources, pursuant to s. 227.19 (4) (b) 2., Stats., requests the Department of Natural Resources to modify clearinghouse rule 03-017 by creating an additional exception to the prohibition on feeding wild animals in proposed s. NR 19.60 (1). Pursuant to the additional exception, feeding of wild animals would be allowed if the feeding meets the following conditions:

--Feed may only be placed north of state highway 29.

--The authorization to place feed does not apply in areas north of state highway 29 established by the department of natural resources as a CWD eradication zone after the effective date of CHR 03-017. The department may by rule ban feeding in an intensive harvest zone or herd reduction zone north of state highway 29 that is established after the effective date of CHR 03-017. A ban on feeding under this paragraph does not apply until at least 72 hours have elapsed following notice by the department of the ban in a newspaper likely to give notice in the area.

--Feed must be placed by, or on behalf of, an individual within 100 yards from the individual's primary residence or by the owner or employee of a business that is open to the public within 100 yards from a building containing the business.

--Notwithstanding the previous paragraph, feed may not be placed within 100 yards from any highway that is posted with a speed limit of at least 45 miles per hour.

--The total amount of feed placed at any time may not exceed 2 gallons per residence or building.

Motion on baiting wild animals

MOVED, that the Senate Committee on Environment and Natural Resources, pursuant to s. 227.19 (4) (b) 2., Stats., requests the Department of Natural Resources to modify clearinghouse rule 03-017 by creating an additional exception to the prohibition on baiting wild animals in proposed s. NR 10.07 (2). Pursuant to the additional exception, baiting deer would be permitted during any open season for deer hunting, in the area to which the open deer season applies. The total amount of bait placed at any time may not exceed 2 gallons per hunter. The exception would not apply in an area established by the department of natural resources as a CWD eradication zone, intensive harvest zone or herd reduction zone unless the department authorizes, by rule, the use of baiting for deer hunting in one or more of these zones.

Motion to prohibit shooting deer from aircraft

MOVED, that the Senate Committee on Environment and Natural Resources, pursuant to s. 227.19 (4) (b) 2., Stats., requests the Department of Natural Resources to modify clearinghouse rule 03-016 to delete all provisions in the proposed modifications to s. NR 10.07 (1) (a) that permit shooting deer from an aircraft.

Baiting/Feeding Compromise Brainstorms

One or two year sunset of current proposal (DNR would like this)?

1. Is it possible to separate baiting and feeding in a compromise?

    - No feeding during deer hunting season if baiting not allowed

2. Volume (two-gallons sounds like prevailing theory, but not enforceable on public land).

    - Limit two two-gallons per forty in contiguous ownership?

3. Type of material (manufactured or raw products)

4. Where can bait be located

    Distance from paved roadways (100 yards is DNR suggestion)?
    Feed should be located within 50 yards of a home if baiting not legal.
    Public v. private lands?

5. Which geographic areas

    - HWY 10 – Stevens Point
    - HWY 29 – Wausau
    - HWY 64 – Merill
    - County and counties surrounding locations where CWD has been identified in wild or captive herds (DNR prefers this, has some scientific merit).
    - No baiting/feeding in Manitowoc County (Sen. Leibham).

6. When should feeding be allowed?

    - No feeding during deer season if we say no baiting?
    - Automatic end to baiting and feeding if CWD or TB found outside of zones (DNR suggestion).

6. Who can bait

    - DNR board members would like to allow disabled permit holders to bait if there must be a compromise.

7. Senator Schultz aerial shooting concerns (KENZIE WILL HANDLE)

8. Richland County concerns (JOHNSRUD WILL HANDLE)

- BANNED IN "HOT SPOTS"
- ~~BAIT~~ FEED NO MORE THAN 2 GALLONS ON THE GROUND AT ANY GIVEN TIME
- ~~BAIT~~ FEED MAY NOT BE PLACED WITHIN 100 YDS ~~OF A~~ OF HIGHWAY (45 MPH +)
- FEED MUST BE PLACED WITHIN 100 YDS. OF AN OWNER OCCUPIED ~~HOME~~ RESIDENCE

FEEDING

Baiting/Feeding Compromise Brainstorms

One or two year sunset of current proposal (DNR would like this)?

1. Is it possible to separate baiting and feeding in a compromise?

    - No feeding during deer hunting season if baiting not allowed ie. Sept. 1 – Dec. 1

2. Volume (two-gallons sounds like prevailing theory, but not enforceable on public land).

    - Limit two two-gallons per forty in contiguous ownership?

3. Type of material (manufactured or raw products)

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    Distance from paved roadways (100 yards is DNR suggestion)?
    Feed should be located within 50 yards of a home if baiting not legal.
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6. Who can bait

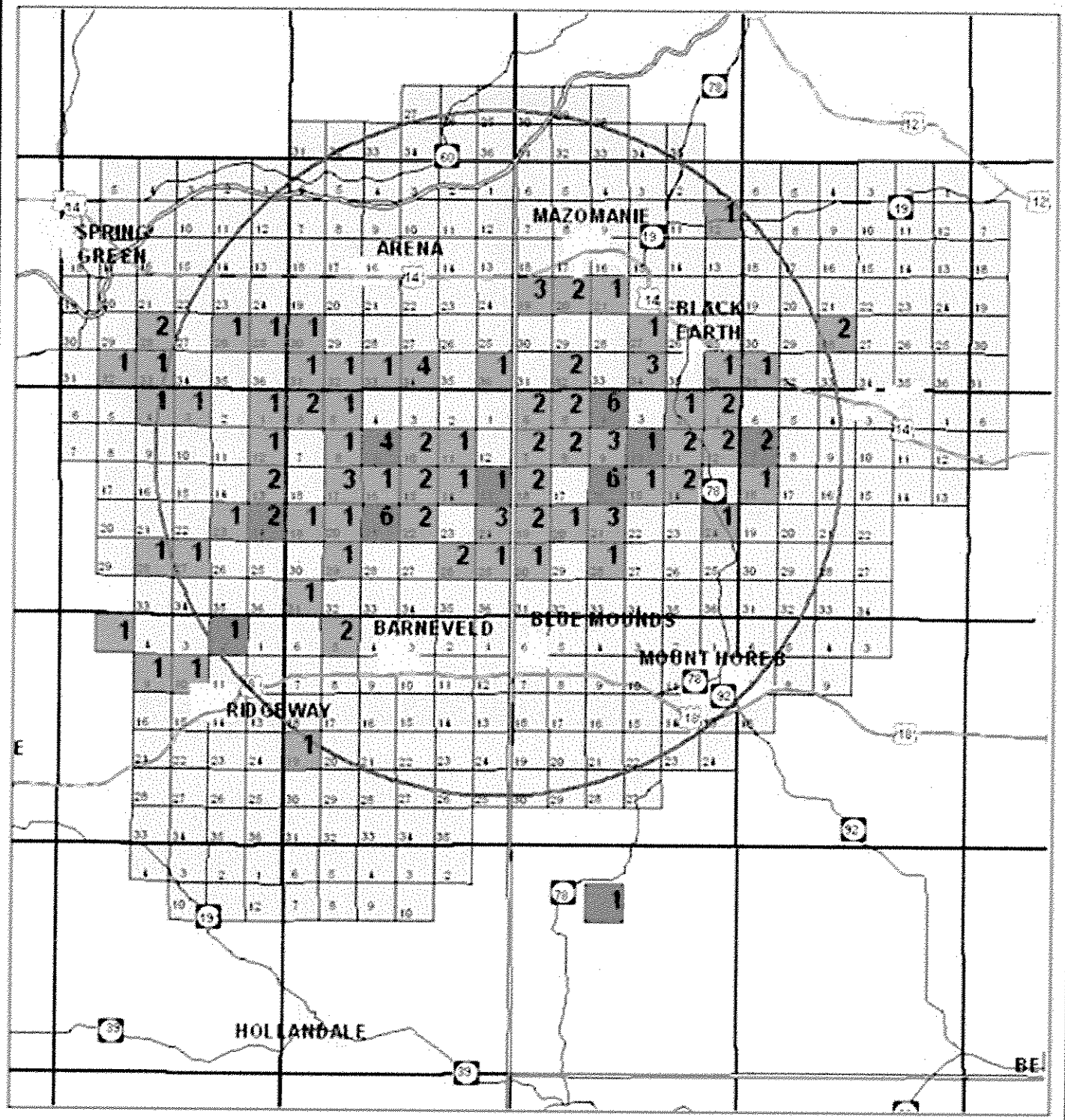
    - DNR board members would like to allow disabled permit holders to bait if there must be a compromise.

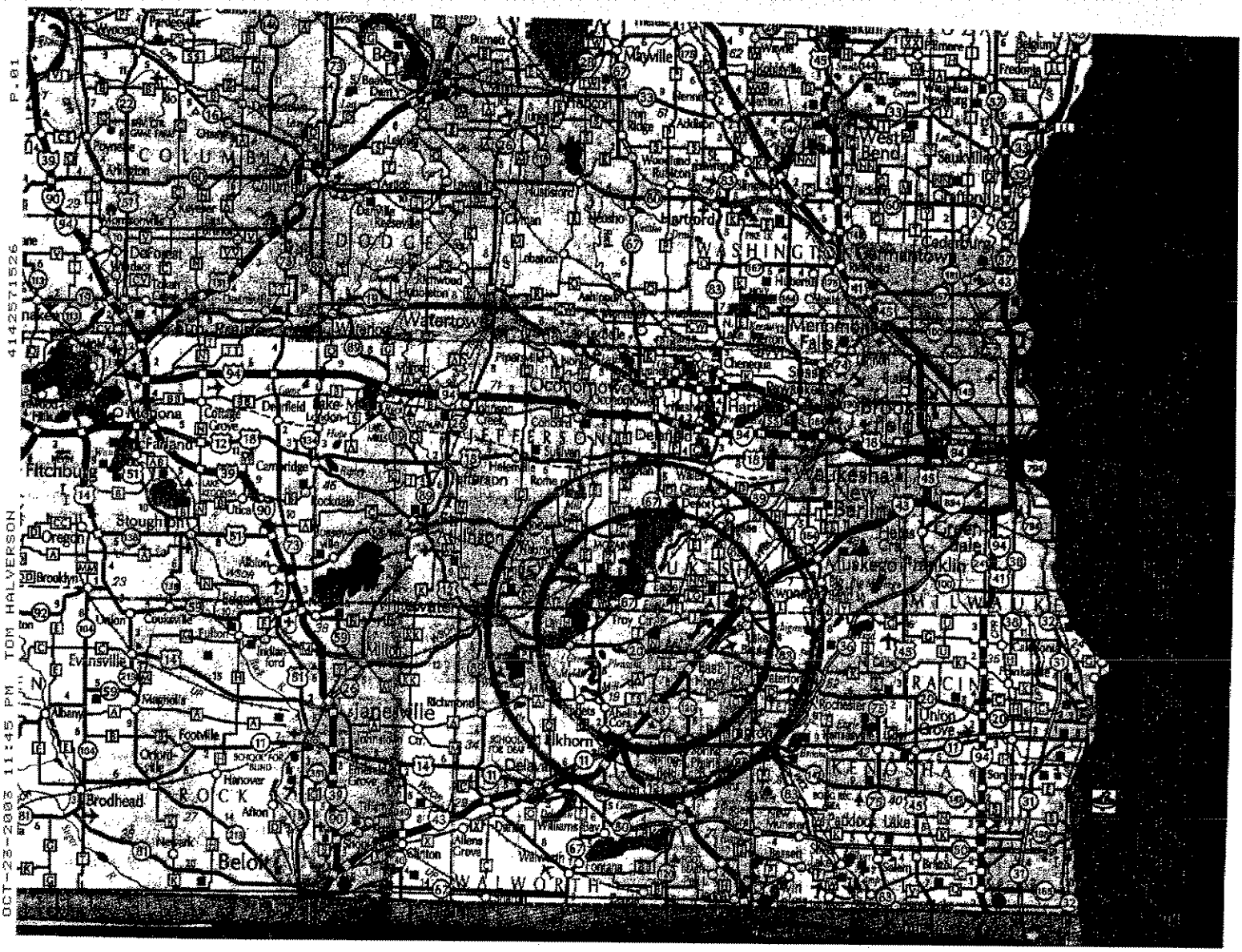
7. Senator Schultz aerial shooting concerns

8. Richland County concerns

Feeding goes off in June

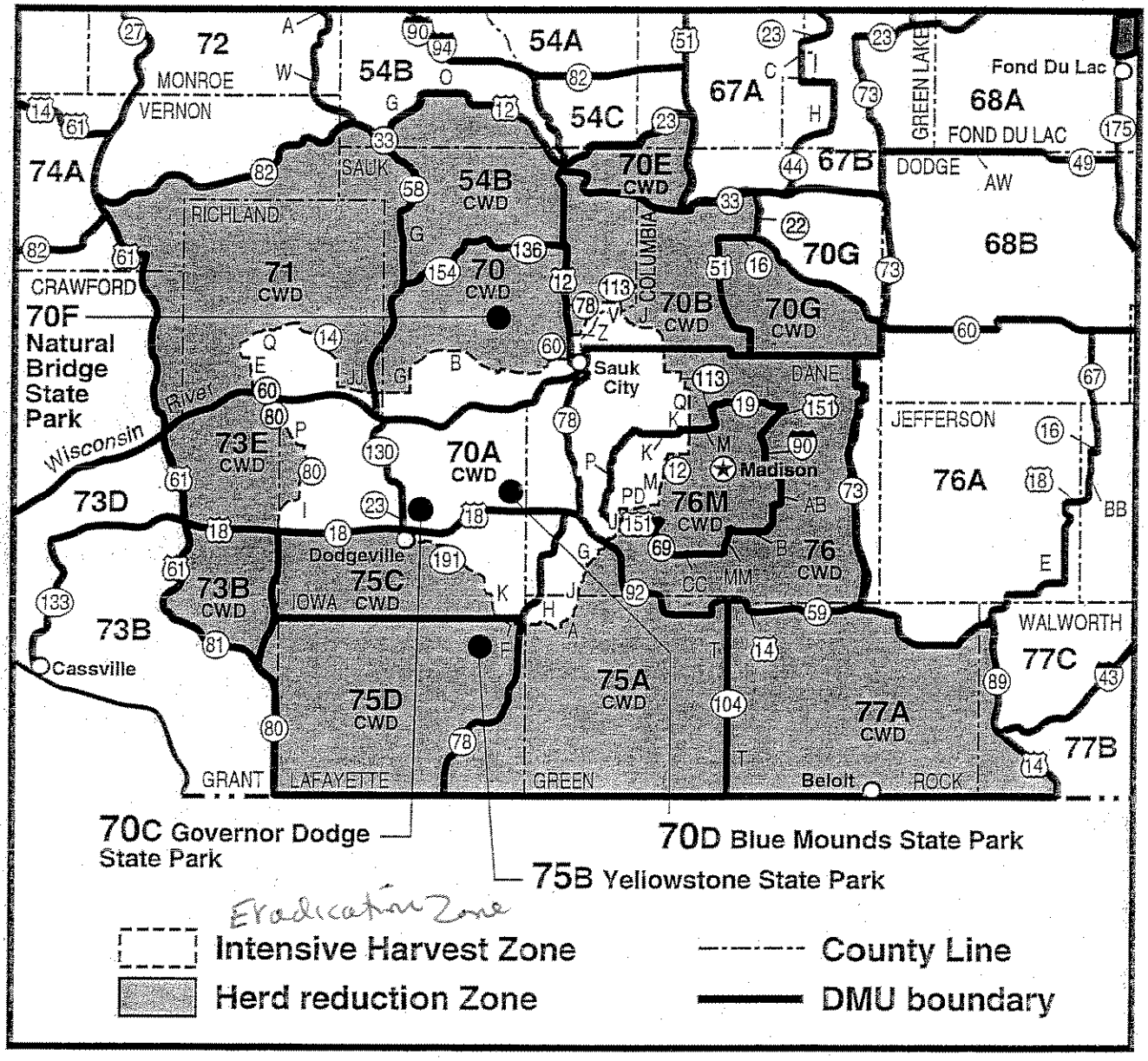
Chronic Wasting Disease Eradication Zone (4/11/03)





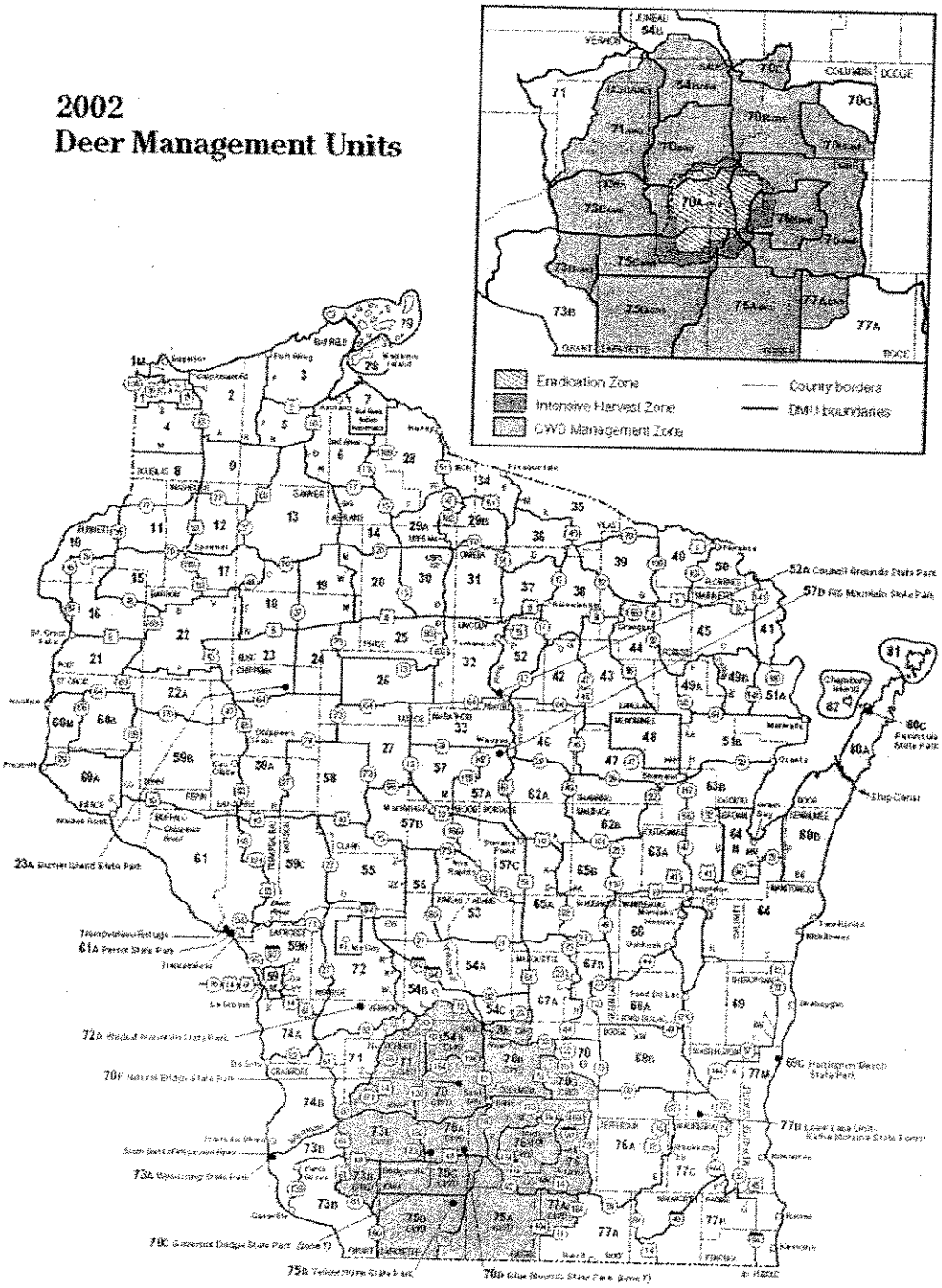
Section 31. NR 10.28(3) is created to read:

NR 10.28(3) CWD HERD REDUCTION AND INTENSIVE HARVEST ZONES.



APPENDIX A: 2002 DEER MANAGEMENT UNITS

CWD area (shaded) and outstate area (not shaded)



Possible Modifications to CR 03-016 and CR 03-017

Clearinghouse Rule 03-016 – CWD management, eradication zones, etc.

Request modification to delete references to "hunting or shooting from aircraft" under Section 21, NR 10.07(1)(a)1 and 2 **(Sen. Schultz/Rep. Freese support)**

Clearinghouse Rule 03-017 – ban on baiting and feeding

Rule Sunsets on June 30, 2004 (allows full Legislature to take up the issue)

With the following possible modifications:

No baiting and feeding allowed on public lands

Request modification to NR 10.07(2)(b) to include the following Exceptions:

No more than 2 gallons of bait within any 20 acre site may be used in areas north of Highway 29 provided the bait is placed in a manner that allows for its removal daily at the end of hunting hours for deer established in NR 10.06(5).

-OR-

No more than 2 gallons of bait with any 20 acre site may be used in areas north of Highway 10, with the exception of Manitowoc, Brown, and Calumet counties, provided the bait is placed in a manner that allows for its removal daily at the end of hunting hours for deer established in NR 10.06(5).

-OR-

No more than 2 gallons of bait may be used in areas north of Highway 29

-OR-

No more than 2 gallons of bait may be used in areas north of Highway 10, with the exception of Manitowoc, Brown, and Calumet counties

Request modification to NR 19.60(2) to include the following Exceptions:

Feeding of wild animals with no more than 1 gallon of feed per day used in areas north of Highway 29 from January 1 to May 31. Feed must be placed within 100 yards of an owner-occupied residence or business of operation. Feed may not be placed within 50 yards of any highway that is posted with a speed limit of 45 miles per hour or greater.

-OR-

Feeding of wild animals with no more than 1 gallon of feed per day used in areas north of Highway 10, with the exception of Manitowoc, Brown and Calumet counties from January 1 to May 31. Feed must be placed within 100 yards of an owner-occupied

residence, business of operation. Feed may not be placed within 50 yards of any highway that is posted with a speed limit of 45 miles per hour or greater.

Motion

The Joint Committee for Review of Administrative Rules:

1. Extends, pursuant to s. 227.24 (2) (a), Stats., the effective period of emergency rules NR 10 and 15 except for sections 20, 21 and 37 until September 1, 2003.

2. Extends, pursuant to s. 227.24 (2) (a), Stats., sections 20, 21 and 37 of NR 10 and 15 until April 30, 2003.

3. Requests that the Department of Natural Resources amend emergency rules NR 10 and 15 to provide for all of the following:

    a. A person, north of state highway 10, with the exception of Manitowoc County, may not hunt with the aid of more than two gallons of bait within any 40-acre site. BAIT ALLOWED NORTH OF HIGHWAY 10

    b. A person, in order to draw deer or elk for viewing purposes on sites outside of the CWD management zone, may place no more than **two gallons of feed** within 100 yards of a owner occupied residence, except that feed may not be placed within 100 yards of any highway that is posted with a speed limit of at least 45 miles per hour.

STATEWIDE FEEDING

~~BAIT BAN NOT APPLIED IN COUNTIES WHERE CWD~~

~~WAS~~ DEER

A PERSON MAY NOT HUNT WITH THE AID OF MORE THAN TWO GALLONS OF BAIT

Request modification to NR 19.60(2) to include the following Exceptions:

Feeding of wild animals with no more than 2 gallons of feed may be placed in areas north of Highway 29. Feed must be placed within 100 yards of an owner-occupied residence and may not exceed 2 gallons at any given time. Feed may not be placed within 100 yards of any highway that is posted with a speed limit of 45 miles per hour or greater.

[redacted] RULES

DNR: TOM HAUGE

ACT 108 AUTHORIZED REGS. ON FEEDING TO JUNE, 2004

DATCP: DR. SHELBY MOLINA, CWD MONITORING COORDINATOR

SUPPORT BOTH RULES

BAIT/FEED CAUSES CLEAR TRANSMISSION OF DISEASES

IS TRANSMISSIBLE FROM ANIMAL TO ANIMAL

RESEARCH IS SLOW AND $$ IS TIGHT TO RESEARCH

PREVENTATIVE MEASURES NEED TO BE TAKEN NOW

WCA = WI CATTLE ASSOCIATION

DONNA WHITE, STATE SNOWMOBILE ASSOC. (ASSOC. OF WI SNOW. CLUBS)

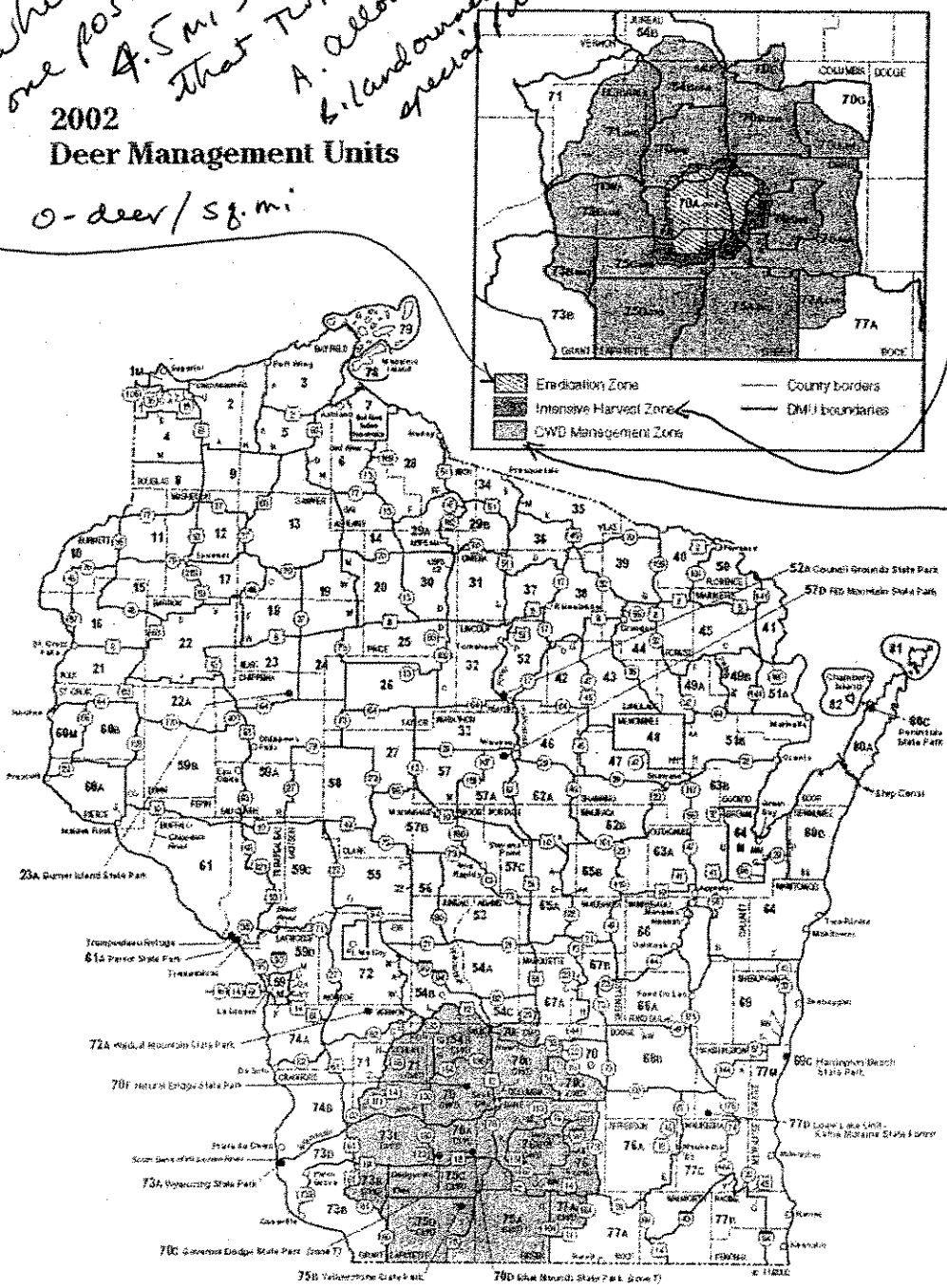
JCRAR COMPROMISE? 2 GALLON PER DAY IN A 10 x 10 AREA, NO. OF HUNTING

BEER 2K COMPROMISE?

DNR SAYS COMPROMISES ARE VERY DIFFICULT TO ENFORCE

APPENDIX A: 2002 DEER MANAGEMENT UNITS

CWD area (shaded) and outstate area (not shaded)



FEEDING COMPROMISE

Feeding of wild animals with no more than 2 gallons of feed may be placed in areas north of Highway 29. Feed must be placed within 100 yards of ~~primary residence~~ (or business) and may not exceed 2 gallons at any given time. Feed may not be placed within 100 yards of any highway that is posted with a speed limit of 45 miles per hour or greater.

buildings open to the public

BAITING COMPROMISE

A person may not hunt deer with the aid of more than two gallons of bait.

NEWLY CREATED CWD ZONES

If the DNR creates another CWD zone – regardless of location in the state – both baiting and feeding will be banned unless DNR grants a permit to the landowner

HUNTING FROM AIRCRAFT

Request modification to delete references to "hunting or shooting from aircraft" under Section 21, NR 10.07(1)(a)1 and 2

idea
4.5 mi. radius from location of identified CWD. Class A notice w/ map. w/i 72 hrs. As of publishing date.

Legislative Update

by: State Senator Neal J. Kedzie
11th Senate District

Natural Resources Rules Gain Statewide Attention

There's no doubt this is a busy time of year for the Legislature. Much of our time is focused on balancing the biennial budget, however the state doesn't stand still while the budget is being addressed. In fact, two natural resource rules have come before the Senate Committee on Environment and Natural Resources that are generating a great deal of attention. As Chair of the committee, I take a great interest in both the proposed Chronic Wasting Disease (CWD) rule - which among other things prohibits baiting and feeding of deer - and the Air Emissions rule.

CWD has changed the face of deer hunting and herd management in Wisconsin. In response to this deer health crisis, the Department of Natural Resources (DNR) instituted an emergency baiting and feeding ban last year. That ban has since lapsed and the DNR is now asking for a permanent ban until CWD can be contained.

In the past year, the DNR has tested about 40,000 deer, and the disease has not been found in any wild deer outside of southwest Wisconsin, though it has been found in some game farm animals. Because CWD is similar to other brain diseases found in sheep and cattle, researchers believe the disease is spread through close contact, and that congregation around feed piles will serve to increase the rate of spread.

Opponents of the ban say that the ban is not necessary, and is doing harm to both the economy and their sport. They argue there is no evidence the disease is spread by baiting or feeding. Some hunters contend that in certain areas of the state, baiting is important because without the bait piles, deer will starve, and hunters will be unable to harvest an adequate amount of deer. Further, they are concerned that the state's economy is being damaged due to the loss of business to feed mills because of the ban, and likely loss in license fee revenues.

-more-

Proponents of the ban believe current testing is not conclusive regarding the spread of CWD thus extending the ban only makes sense until more is known. Further, if the baiting and feeding ban helps contain the spread of CWD, the precaution will have been worth the short-term inconvenience and economic losses. Lifting the ban without more information would jeopardize not only the health of the deer herd in the state, but could cause a spread which would threaten other states, and potentially livestock.

The issue has raised interest and emotions statewide. A legislative committee recently passed a compromise plan to allow limited baiting and feeding in some areas of the state, but the Natural Resources Board rejected the plan in favor of continuing the ban. The ban will require legislative review, which will come from the Senate and Assembly Natural Resources committees. As Chair of the Senate committee, I continue to take input from people across the state and will be addressing it at a joint public hearing of those committees on May 14, 2003 here in Madison. The decisions we make now about handling this crisis will have long-lasting effects, and we want to assure our legacy is one of which we can be proud.

Another contentious issue facing both committees this month is a rule to greatly expand the regulation of air emissions. At issue is the number of substances the DNR would like to regulate. Currently the US Environmental Protection Agency regulates 189 hazardous air pollutants and Wisconsin's DNR regulates a total of 438. The items on the DNR list include a range of substances from mercury to flour dust.

By this rule, the DNR is asking to add 260 more substances to their list, bringing the total number of substances it regulates as air pollutants to 578. That would make the DNR program three times more encompassing than the federal program. Critics of the rule question whether the DNR can or should regulate so many more materials that the EPA does not deem hazardous. Further, many Wisconsin business believe this rule would greatly increase their cost of doing business and create another significant layer of regulatory burden. And some have questioned the DNR's ability to regulate new emissions while it struggles to permit and regulate the current emissions rules.

-more-

Regardless of your position on either rule, the process by which those rules move through the Legislature offers the opportunity [illegible] The hearing for the CWD rule is set for May 14, 2003 and [illegible] be May 22, 2003, both here in Madison at the Capitol. If you would like more information, you may contact my office at any

Sen. Kedzie can be reached in Madison at P.O. Box 7882, Madison, WI 53707 or by calling toll-free 1 (800) 578-1457. He may be reached in the district at (262) 742-2625 or on the World Wide Web at www.[illegible].gov

-30-

Identify feeding station

High Penalty
Limited Amount of feed

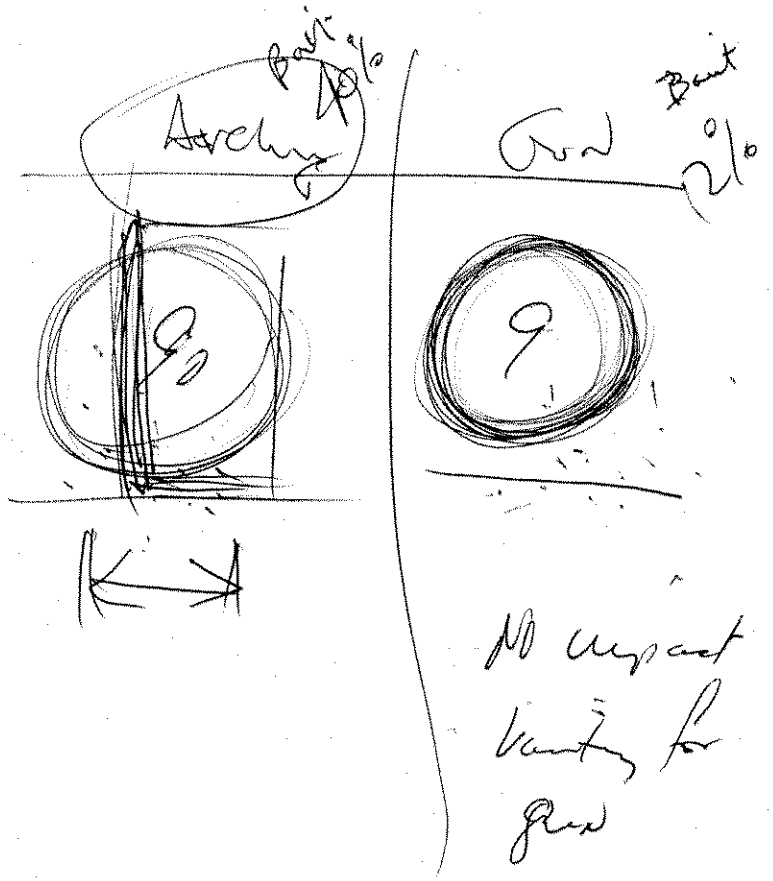
Civil Rule

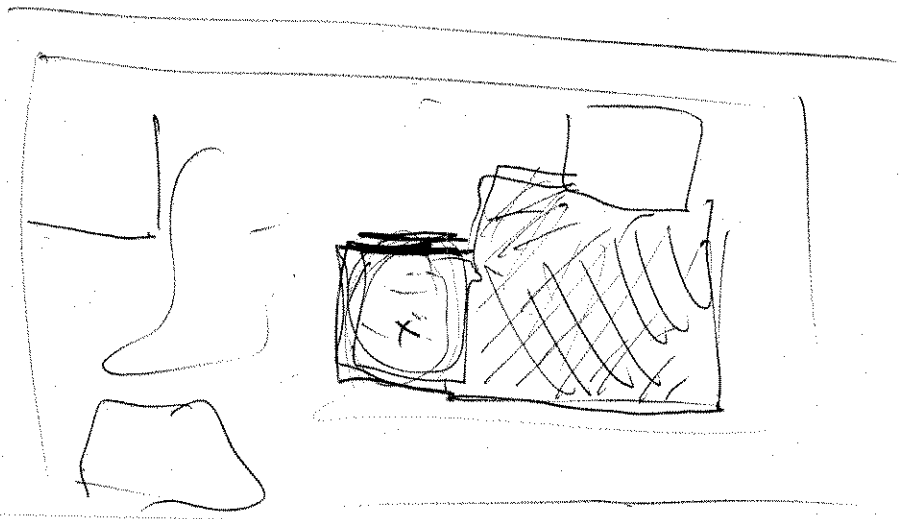
1 yr. sunset on rule. June 30 2004

Need extension by legislature

(1) No shooting from helicopters (NIO

(2) Modify drawing Richland Co. from Eradication





Compromise Issues

- Amount of feed (2 gal)
- location at same site vs. moving it
- Bleaching of ground
- Baiting - remove daily?
- Disabled hunters
- If disease found shut down area immediately.
- Penalties

2 gal ~ 10 ft/day

...y needs to be a...
...ill meet the nation's grow...
...to bridge the gap between
...combination of energy efficiency and
...eased domes... production. Right now, neither the House
...nor the Senate bill meets those criteria.

—Milwaukee Journal Sentinel

Scrap ethanol subsidies

This week, the Senate embarked on one of its periodic debates over ethanol—the politically blessed motor fuel derived from corn, which is added to gasoline. Supporters are trying to expand the program, but instead it should be ended. Ethanol subsidies aren't about energy. They're about politics.

The ethanol program authorizes a 53-cent-per-gallon tax break, allowing the fuel to compete on the market with straight gasoline. At issue is whether production under the program should be increased to 5 billion gallons, compared with the current 2.13 billion gallons.

Supporters say greater use of ethanol would help reduce greenhouse gases and ease U.S. energy dependence. There are problems with both these claims.

Ethanol does produce less carbon monoxide, but it pollutes in other ways. It releases more nitrogen oxide, a key ingredient in smog, and it evaporates faster. Some studies show that ethanol-laced gasoline is harder than straight gasoline to remove from waterways after spills. And ethanol plants have been cited by the government as significant sources of pollution.

Even ethanol's value as an energy source is in doubt, given that it takes a lot of energy from coal, oil or natural gas to make the stuff. Some experts say more energy is consumed in producing ethanol than it releases when burned; other experts cite studies showing a net gain.

But it's revealing that this is even an issue. We don't have similar arguments about gasoline; there's no question about its energy value.

When you include the oil in the fertilizer needed to grow the corn, it's hard to justify ethanol's value as an energy source, especially since the subsidy translates into a billion-dollar loss for the highway fund every year. Several years ago, an Agriculture Department study concluded that without the subsidy, ethanol producers wouldn't even survive.

The program should be eliminated, not expanded. But if the past is any guide, none of these considerations will make much difference. The measure before the Senate has bipartisan support because a lot of corn is grown in political swing states. This is a politics program, not an energy program.

—The Kansas City Star

Hunters discover worst enemy

Front-page photographs last week featured burly men in full-dress combat gear grinning broadly while holding up the limp, dead bodies of small birds.

JOEL McNALLY

The large men were gleefully celebrating what they thought was a glorious victory. The state's first dove hunting season was a symbolic triumph, they proclaimed, against wimpy, anti-hunting forces in Wisconsin.

It was symbolic, all right. Their beaming photographs made excellent recruiting posters for People for Ethical Treatment of Animals.

Large beasts crashing around Wisconsin's woods while joyously blowing mourning doves out of the sky are like aging dinosaurs destroying their own habitat and hastening the day of their own extinction.

Oversized bullies don't attract many friends. The Bush administration learned that when it ignored the feelings of the rest of the world to invade and occupy another country just because it could. Now it's having a tough time getting other countries to help clean up the mess.

Shoving a dove hunt down the throats of Wisconsin just because the hunting lobby is big enough and crude enough to want one has the same effect of swelling the ranks of anti-hunters.

No less a pro-hunting politician than former Gov. Tommy Thompson was smart enough to understand that. That is why Thompson opposed a dove hunt. He said hunters were going to need nonhunting friends and that blowing away the bird of peace wasn't going to win them any.

But when anti-hunting groups successfully stopped the launching of a dove hunt two years ago with a court injunction, hunters went a little crazy. Hunters had never been denied anything politically in the state of Wisconsin before.

They'd successfully transformed the Department of Natural Resources into the Department of Nuts with Rifles. All they had to do to get the DNR to launch a new hunting season was for hunters themselves to vote for it in their annual "conservation" meetings. But now a court was actually telling hunters they couldn't shoot anything they wanted. The injunction infuriated hunters who otherwise wouldn't have bothered to hunt a small bird with about as much meat on its bones as a large, flying insect.

Suddenly, hunters were determined to blow away every mourning dove in Wisconsin that so much as cooed.

What really angers hunters isn't bird lovers. It's that bird lovers and other intellectuals who exalt the beauty of nature don't love hunters. Hunters resent being treated like they are old and embarrassing just because, well, they are old and embarrassing. LL Bean and other hip catalogs sell expensive designer outfits and gear for well-educated nature lovers to take into the unspoiled wilderness for some kind of spiritual communion.

But none of those catalogs show anybody killing anything and gutting it. Communing with nature is far too refined and aesthetic an experience to be defiled with lowlife blood sports.

Some states have laws against hunters firing guns within sight of a highway. Contrary to popular belief, this is not a safety regulation. These hunters are required to be much farther from traffic than a bullet could ever travel. The law is to protect motorists from the unpleasant sight of packs of grown men blowing away animals.

Wisconsin hunters brag about introducing the next generation to the hunt just as their fathers did for them. But how many of those kids share with their college classmates the bloody killing grounds of their fathers and grandfathers?

Dove slaughter is right up there with deer baiting as an ugly little activity that is not mentioned in polite society. The concept behind deer baiting is to take all that bothersome hunting out of hunting.

All you have to do is lay out a lavish banquet for deer and invite a whole herd over for dinner. Then, at some point when everybody is feeling a little groggy after cocktails and a big feast, you jump out from behind a bush with a gun and massacre the whole party.

Whenever you go into a bar and see a deer's head on the wall with Christmas lights strung through the antlers and a funny hat, you know some deer baiter plugged that deer while it was being the life of the party with a bunch of deer buddies.

Most legislators pay a lot more attention to hunters than nonhunters. That's why they've refused to extend the ban on deer baiting the DNR imposed to try to prevent the spread of chronic wasting disease.

But the more hunters flex their political clout and flaunt their most boorish behavior, the faster they are hastening the political backlash.

Wisconsin's biggest anti-hunting movement has no connection to PETA. It's the action of private property owners all over the state to put undeveloped land off-limits to hunters.

While all those dove hunters were gloating and proudly holding aloft their tiny prey, a lot more signs went up.

Large beasts crashing around Wisconsin's woods while joyously blowing mourning doves out of the sky are like aging dinosaurs destroying their own habitat and hastening the day of their own extinction.

Joel McNally is a syndicated columnist. His e-mail address is jmcnally@wi.rr.com.

EDITORIAL

Ban on baiting comes just in time

With bow hunters returning to the woods in search of whitetails Saturday, we're glad the Department of Natural Resources Board did the only expedient thing it could—ban baiting and feeding of deer in 22 southern Wisconsin counties.

The board approved the ban, which includes Rock, Walworth, Green, Jefferson and Dane counties, in an effort to slow the spread of chronic wasting disease. The ban covers any county within 10 miles of where a deer was discovered with the disease. It took effect Thursday, just two days before bow season began; gun seasons start Oct. 30.

The board bent to political reality after shortsighted lawmakers last month shot down the DNR's desire for continuing a statewide ban. Lawmakers saw a statewide ban as too restrictive, bowing to the equally shortsighted pleas of animal lovers, feed companies and those hunters who'd rather sit over bait piles than pursue deer in a more sporting and traditional manner.

Unfortunately, a coalition of five groups of hunters, landowners and wildlife enthusiasts urged a boycott of this fall's deer hunt Wednesday to protest the DNR Board's decision. Like lawmakers, such protests are shortsighted and misguided.

A legislative committee rejected the blanket ban only days before a new study out of Colorado revealed that the contagious and fatal brain disease is spread more easily than previously thought. The study analyzed two populations of captive mule deer. One set was born-to-captivity does that had contracted the disease. The second was born-in-the-wild deer free of the disease but later captured and placed with the first group. All of the animals eventually contracted the disease, discounting the theory that maternal transmission was key to its spread.

Lawmakers still could have rejected the DNR board's emergency rule, expected to last 150 days. However, they are working on a bill that would limit baiting north of Highway 54 during deer season to two gallons per 40 acres. That proposal also would allow deer feeders to set out two gallons per day within 50 yards of an owner-occupied residence statewide except in areas labeled as disease eradication or management zones.

Outside of these even more unpalatable kill zones, bans on feeding and baiting look like the most logical defense against the spread of chronic wasting disease. They are also a defense against the potential for a devastating outbreak of bovine tuberculosis in the deer herd, which could transmit it to cattle. Michigan officials suspect deer baiting caused a bovine TB outbreak there.

Because of both concerns, a statewide ban on baiting and feeding is preferable. But lacking that option, the Wisconsin DNR board took the only step it could.

Help out in Walworth County

We're also pleased with the DNR's new plan to step up surveillance of Walworth County deer, testing as many as possible after a doe in a Fontana park was found to have chronic wasting disease.

Officials had considered the county to be a higher risk area because the disease was found in deer killed just across the border in Illinois. Before the doe was found in Fontana, two miles outside an area established to control spread of the disease, wildlife officials had planned to limit testing to whitetails in an area near a game farm in Troy Township where six deer had tested positive.

Hunters offering their deer heads to the state for testing will get the results in a matter of weeks instead of months because the DNR will use new rapid tests. We urge hunters in Walworth County to take advantage of testing and help wildlife officials gain as much knowledge as possible to prevent further spread of the disease.

Could environmental contamination be a possibility of causing the CWD condition?

The town of Vermont used to mine lead. CADMIUM is a byproduct of mining lead.

As early as the Civil War time, they mined lead and made lead balls in the Mt. Horeb area.

What did they do with the WASTE from mining lead in the past?

LAB NUMBER: 84481
SAMPLE ID: K7

REPORT OF ANALYSIS

DATE SAMPLED: NONE GIVEN
DATE RECEIVED: 1/14/03 9:30 AM
DATE REPORTED: 1/17/03 PAGE: 7

PARAMETER	RESULT	UNIT	DETECTION LIMIT	ANALYST	ANALYSIS DATE	METHOD REFERENCE
Sample Digestion	0			MG	1/15/03	SW846-3050B
Cadmium	2.32	mg/kg	0.10	KDK	1/16/03	SW846-6010B
Copper	14.23	mg/kg	0.10	KDK	1/16/03	SW846-6010B
Magnesium	10284.6	mg/kg	1.0	KDK	1/16/03	SW846-6010B
Phosphorus	1315	mg/kg	1	KDK	1/16/03	SW846-6010B
Zinc	86.46	mg/kg	0.10	KDK	1/16/03	SW846-6010B

Soil sample from farm field down hill from same highway in Southern Wis

Cadmium should be .5 mg/Kg

Magnesium should be 90-140 mg/Kg not 10284.6

LAB NUMBER: 64475
SAMPLE ID: KR

REPORT OF ANALYSIS

DATE SAMPLED: NONE GIVEN
DATE RECEIVED: 1/14/03 9:30 AM
DATE REPORTED: 1/17/03 PAGE: 1

PARAMETER	RESULT	UNIT	DETECTION LIMIT	ANALYST	ANALYSIS DATE	METHOD REFERENCE
Sample Digestion				MG	1/15/03	SW846-3050B
Cadmium	0			KDK	1/16/03	SW846-6010B
Copper	2.36	mg/kg	0.10	KDK	1/16/03	SW846-6010B
Magnesium	9.15	mg/kg	0.10	KDK	1/16/03	SW846-6010B
Phosphorus	55823.0	mg/kg	1.0	KDK	1/16/03	SW846-6010B
Zinc	391	mg/kg	1	KDK	1/16/03	SW846-6010B
	43.67	mg/kg	0.10	KDK	1/16/03	SW846-6010B

Soil sample was taken in a ditch along the highway in southern Wis

Trace element (nutritional) theory of "mad cow" disease:

Murray McBride, mbm7@cornell.edu

The outbreak of "mad cow" disease (BSE) in Britain had been connected epidemiologically to feeding of concentrates containing meat-bone meal (MBM) to dairy calves (Wilesmith et al.,1988, 1991,1992a, 1992b) . Beef cattle breeding herds (which generally do not have their rations supplemented with protein concentrates) have had a much lower BSE incidence in the UK, supporting this feed additive theory of BSE.

Since the practice of feeding animal protein is not new, the disease outbreak in the mid 1980's cannot be explained solely by the hypothesis of a disease agent in MBM. However, it has been hypothesized that the cessation of hydrocarbon solvent extraction of fat from MBM in the early 1980's at most rendering plants in England could have allowed an infective scrapie-like agent to pass into dairy feed (Wilesmith et al., 1991).This may not be the only possible explanation, as it was pointed out by Rhodes (1997) that another change in the cow's diet also could explain the epidemic, which was initiated by the higher prices for imported soy and fish meal in the early 1980's. This forced farmers to shift to greater use of the cheaper MBM. There is also reason to believe that the infective prion, reputed to be the cause of BSE, is sufficiently hardy that changes in the rendering plant processing of MBM may not have greatly affected infectivity. Taylor (1998) stated that "most of the rendering procedures used to manufacture meat and bone meal (MBM) throughout the European Union have been found to be incapable of inactivating BSE and scrapie agents". Even autoclaving at 132-138 C is not completely effective (Taylor, 1998). This observation, along with the fact that other countries have fed similarly processed MBM to dairy animals without causing an epidemic of BSE, suggests that an environmental or nutritional factor in certain regions of the UK is a predisposing or causative factor in the disease.

It is curious that the geographic occurrence of "mad cow" disease (number of cases per 1000 head) is not evenly or randomly distributed in the UK, but has tended throughout the epidemic to be highest in the southern and eastern counties (Wilesmith et al., 1992a). Several counties in this region are known to have widespread copper defiencies in soils and crops (Thornton and Webb, 1980). These crop deficiencies could lead to copper deficiency in ruminants, a fairly well-recognized disease with specific symptoms, in those regions without copper supplements in rations. BSE has tended to have higher occurrence in particular herds, even though there is no definitive evidence that the disease can be transmitted animal-to-animal.Since consumption of MBM presumeably varies from animal to animal, the impact on some animals could be much greater than on others.

One impact of a high-MBM diet could be to induce Cu deficiency, as feeds rich in protein , particularly soluble protein, decrease the efficiency of Cu absorption by ruminants (Rehbinder and Petersson, 1994 ; McDowell, 1985). One would expect heat-processed MBM to be high in soluble protein. Also, animal protein is high in sulfur content, and diets with as little as 0.4 % S can contribute to Cu-deficiency and even cause polioencephalopathy. (The presumed high Fe content of MBM due to blood also has a potentially negative effect on Cu availability). Another is the introduction of Pb, Cd and other toxic metals from organs and bone tissue of diseased "downer" cattle into the feed. On the basis of chemistry, the presence of soluble high-S protein in the rumen might solubilize lead from bone and other tissue, making the lead more bioavailable. The lead concentration in bone tissue of cattle is high in contaminated areas (Milhaud and Mehennaoui, 1988). A similar argument suggests that high-S protein diets could mobilize certain toxic metals from the substantial quantity of contaminated soil that resides in the abomasum of cattle grazing contaminated land.(I recall a situation in which horses and cows were grazing lead-contaminated pasture, with only horses showing clinical signs of lead poisoning. However, when a chelating agent was administered to a cow from the pasture, she quickly developed lead toxicosis).

The use of animal protein, which increases nitrogen in the feed, could lead to a deficiency of essential fatty acids in the cell membranes, reducing membrane integrity , and making the animal more susceptible to encephalomalacia (Crawford et al., 1991). The fairly recent increased use of canola seed cake in animal rations in the UK could also contribute to this nutritional imbalance, as canola has a high sulfur content and can accumulate certain toxic metals from soils in the seed. Feeds which are high in molybdenum relative to copper are well-known to induce copper deficiency in ruminants (McDowell, 1985), and legume forages or soybean meal can have unacceptably high molybdenum content if grown on non-acid soils with more than 3 ppm molybdenum (McBride, unpublished data). This disease is referred to as molybdenosis, and is generally recognized by obvious symptoms such as changes in hair coat pigmentation.

Based on the number of references that can be found where Cu deficiency has been diagnosed in numerous ruminant species in the wild, as well as captive or in a farming environment, Cu deficiency appears to be common. It has been observed in cattle, moose, red deer, Sika deer, elk , muskoxen and goats (Mackintosh,1998 ; Stafford, 1997; Blakley et al.,1998; Arnhold et al., 1998; Gogan et al., 1989), particularly in cases where wild animals have been captive or confined. Wapiti (elk) may be particularly susceptible to Cu deficiency, and the disease is reported frequently in red deer on farms (Blakley et al., 1992). Confining wild ruminants on farms appears to increase the risk of certain diseases, including copper and other trace element deficiencies (Mackintosh, 1998). Wild ruminants may be able to compensate for soil deficiency of particular micronutrients by obtaining a more varied diet than confined ruminants restricted largely to grass forage (Stafford, 1997). Interestingly, wild ruminants appear to be better adapted to low-Cu diets than most domesticated ruminants, as the necessary level of Cu in the liver tissue of domesticated sheep and cows (35 mg/kg dw) is higher than that for deer (10-20 mg/kg) (Arnhold et al., 1998). Domestic goats require even less Cu (8 mg/kg in liver), and it is interesting that the Cu level in the cerebrum is a more reliable indicator of Cu deficiency in goats than that in the liver (Arnhold et al., 1998). Cerebrum Cu concentrations in goats are generally less than 10 mg/kg (dw), levels considered to be marginal or low in sheep and cattle.

Neurological Symptoms in Copper Deficiency and TSE's

In sheep, copper deficiency has been recognized in the UK and elsewhere for a long time as the disease referred to as swayback. Neurological degeneration from swayback has generally been described as demyelination, but more recent investigations of the neuropathology note vacuolation of the white matter, neuronal necrosis, gliosis (Mohammed et al., 1995). Demyelination has been observed in deer with copper deficiency (Geisel et al., 1997; Yoshikawa et al., 1996). However, Yoshikawa et al.(1996) described the neuropathology as "spongy vacuolation and myelin deficiency in the white matter of the spinal cord and brain stem" .

Chronic wasting disease in wild moose has become relatively common in Southern Sweden, and there is evidence that it is caused by Cu deficiency possibly induced by increased molybdenum in the forage (Frank, 1998). Neurological pathology associated with this disease is described as " abiotrophy of the cerebellum characterized by a marked thinning and decreased cellularity of the granular layer and a severe loss of Purkinje cells, leaving empty 'baskets' as reminiscences". (Rehbinder et al.,1991; Rehbinder and Petersson, 1994).

Some researchers believe that neuronal degeneration in a number of diseases could have Cu deficiency as an etiological factor (Hartmann and Evenson, 1992). Menkes' kinky hair disease in infants and young children is a rare X-chromosome-linked genetic disorder of copper transport which appears to result from copper being trapped in certain tissues, especially the kidneys, by abnormal metabolism of metallothioncin (Nooijen et al., 1981; Hart, 1983). This leads to copper deficiency, particularly in the brain, causing irreversible damage. The

neurological degeneration in Menkes' disease is pathologically similar to that in Cu deficiency of sheep (swayback) (Tan and Urich, 1983), evidence that brain damage in Menkes' is substantially due to Cu deficiency. Neurological damage progresses in infants in spite of copper therapy (Johnsen et al., 1991), with copper accumulating in certain tissues incliuding the kidney, and remaining low in the brain and liver.

Some descriptions of the pathology of central nervous system degeneration from Menkes' disease include :

"...neuronal destruction was widespread in the cerebral gray matter and in the cerebellum, and there was associated gliosis. The changes in the cerebellum were particularly severe , with neuronal loss in the internal granular cell layer. Many Purkinje cells were lost...." (Moon et al., 1987)

"... prominent vascular, cerebral and cerebellar degeneration." (Morgello et al., 1988)

"....marked neuronal loss and gliosis in most areas of the cerebral and cerebellar cortices, midbrain , pons and medulla. The spinal cord showed severe demyelination " (Uno and Arya, 1987)

" The cerebellum showed the most striking abnormalities : severe lack of internal granule cells. Purkinje cells with weeping willow pattern..." (Robain et al., 1988)

These descriptions bear a marked similarity to those noted above for Cu deficient ruminants, and, as will be discussed later, have considerable similarity to the neuropathology of the "transmissible spongiform diseases" of animals and humans.

Spongiform change itself does not appear to be particularly unique to prion diseases. For example, lead poisoning in dogs produced a neuropathology described as "cerebrocortical lesions comprising spongiosis, vascular hypertrophy and gliosis", as well as "spongiform changes" in the cerebellum with "spongiosis of the Purkinje cell layer and vacuolation of Purkinje cells" (Hamir et al., 1984). In cattle, Christian and Tryphonas (1971) observed that chronic lead poisoning produced "astrocytic swelling and development of focal status spongiosis" and "neuronal necrosis", and remarked that lead encephalopathy may be difficult to distinguish from polioencephalomalacia (PEM), especially in the acute stages. PEM was initially thought to be a thiamin deficiency, as the administration of thiamin often alleviated symptoms. However, recent evidence suggests that thiamin has the ability to counteract lead toxicosis (Gould, 1998).

Demyelination has been usually associated with Cu deficiency, for example, swayback disease in lambs. However, degeneration of myelin sheaths has also been reported in spongiform CNS disease in goats and mule deer (Obermaier et al., 1995; Guiroy et al., 1993) , as well as in scrapie and Creutzfeldt-Jacob disease (Walis et al., 1997; El Hachimi et al., 1998). Copper deficiency is also associated with neuronal degeneration and spongiform pathology, so again, we see evidence that the neuropathology of these presumed different diseases has similarity that may confuse diagnosis. Treatment of experimental animals with Cu-chelating compounds produces neural abnormalities including "spongiform changes in white matter" and "reduced myelin development" (Tanaka et al., 1993).

Given this unclear distinction in pathological symptoms of TSE's and other CNS diseases, one must question some of the conclusions that have been reached on the occurrence of TSE's in animals where disease transmission studies have not been done. Specifically, the neurological damage caused by Cu deficiency, and possibly exposure to neurotoxins such as lead, may not be easily distinguished from the damage from TSE's.

These unexplained facts would seem to suggest the existence of (as yet undiscovered) location-dependent

environmental factors which may not actually cause TSE diseases, but predispose individuals to infection. For example, Agrimi and DiGuardo (1993) have proposed that the blood-brain barrier may be compromised in susceptible hosts, resulting in localization of metals such as lead in brain tissue, as has been shown in Alzheimer's disease. Heavy metals and/or Cu deficiency may damage the integrity of the blood-brain-barrier, increasing the chance of disease transmission.

Are Heavy Metals a Predisposing Factor in BSE ?

In the UK it is possible that offal from some diseased cattle can contain high concentrations of lead, zinc or cadmium as soils of many regions are badly contaminated by centuries of mining and industrial activity. Thornton and Abrahams (1983) estimated that about 1,000,000 acres of agricultural land in the UK has been seriously contaminated by mining and smelting activities over the centuries, and subsequent dispersal by man and the elements. They found average daily intake of lead (Pb) to be higher than intake of Cu in all herds studied in Derbyshire and Cornwall, with Pb intake more than 10 times Cu intake where soil Pb was higher than 1000 mg/kg. Other references note that while clinical lead poisoning in grazing livestock in the UK is uncommon, subclinical lead poisoning and /or copper deficiency could be having significant and more widespread effects on animal health (Thornton and Webb, 1980) . The rather common copper deficiencies which appear in sheep in the UK (swayback disease) appear to be exacerbated by lead toxicity (Suttle et al.,1975). Animals from some regions of the UK can have high levels of Pb in bone and other tissues, as well as high Cd in kidneys and liver. It is reasonable to assume that many of the "downer" cattle in the UK, which end up in MBM, will have higher than average concentrations of these toxic metals in their tissues and organs.

Contaminant metal binding to PrP in MBM may convert the PrP to the infective form; that is, a form that is not readily digested by proteases in the digestive tract, and that is able to cross membrane barriers into the blood stream and finally to the central nervous system (CNS). A recent study has shown that Cu ions can convert PrP to the infective disease form (McKenzie et al.,1998). Warren (1974) noted a long time ago that there was at least circumstantial evidence for a role of environmental lead in numerous CNS diseases. He pointed to evidence that suggested divalent metal cations alter membrane permeability, and that "heavy metal cations stimulate degradation of the phospholipids in membranes". Since copper deficiency leads to developmental abnormalities in the cerebellum and demyelination of the spinal cord in ruminants (Rehbinder and Petersson, 1994), and recently has been shown to bind with high specificity to PrP, one should also consider that copper deficiency or excess toxic metals might predispose animals to infection with TSE diseases.

Chronic Wasting Disease in Wild Ruminants- a TSE or Cu deficiency?

The above-described effects of feed quality on Cu status in ruminants could perhaps explain the incidence of "chronic wasting disease" in zoo animals and elk and deer confined on ranches. Kirkwood et al.(1993) reported incidence of spongiform encephalopathy in 5 of 8 greater kudu born since 1987 in a zoo in London, although 4 of 5 were thought not to have been exposed to feeds containing ruminant-derived protein. Numerous other cases of "spontaneous" TSE have been reported in the literature.

Generally, wild deer and elk have not shown this disease at high levels except in one region of Colorado and Wyoming. Outward symptoms in these animals are loss of body condition (wasting), behavioral changes, excessive drinking and urinating, salivation, incoordination, and tremors. Recent observations seem to put in question the belief that "chronic wasting disease" (CWD) of these wild animals is a prion disease at all. Sika deer on farms showed enzootic ataxia, with neuropathological lesions reported as spongy vacuolation in white matter of spinal cord and brain stem. The disease was attributed to copper deficiency (Yoshikawa et al., 1996) . Moose in Sweden showed ataxia, wasting, and excessive salivation, with neuropathology reported as cerebellum abnormalities characterized by a marked thinning and decreased cellularity of the granular layer

and a severe loss of Purkinje cells. The disease was again attributed to copper deficiency (Frank, 1998)

There are commonly reported incidences of Cu deficiency, diagnosed on the basis of very low blood and liver copper, in many regions of the world. These deficiencies often occur when wild deer, elk and other ruminants are confined on farms or ranches, and it is notable that CWD was observed in confined populations of deer and elk in Western North America for decades prior to the "outbreak" in wild populations of Colorado and Wyoming. There is evidence that confinement prevents animals from browsing for more Cu-rich plant material.

The occurrence of CWD in deer in the Western US, and no report (as yet) of the disease in the East, is consistent with the fact that soils of the West, particularly in the Colorado-Wyoming region and the arid Southwest, are prone to produce forages with high Mo content relative to Cu, potentially leading to Cu deficiency. Alfalfa hay is often fed to deer and elk on farms.Are we able to distinguish a prion disease from Cu deficiency solely on the basis of observations of symptoms in the field, or even cursory examination of brain tissue?

Potential Copper Involvement in Prion Infectivity

This proposed explanation for predisposition to BSE and other TSE's in ruminants , based on nutritional factors, obviously fails to explain the transmission of the disease from infected animals into experimental animals. Recently, however, Ebringer et al. (1997) have proposed that the development of neurospongiform pathology in the brains of experimentally infected animals is an autoimmune response.If this is correct, the validity of many of the reports of BSE transmission under experimental conditions is questionable.

Even if the prion-only theory of BSE proves to be substantially correct, copper and other trace metals may have a key role in controlling infectivity of this molecule. It now appears that the normal prion protein (PrP) of nerve cells in the brain could have a key role in the critical functions of copper in the brain. Recent work shows that copper rapidly and reversibly stimulates endocytosis of PrP from the cell surface (Pauly and Harris, 1998). This could mean that the normal prion acts as Cu sink, since it strongly chelates the metal, or functions as a carrier to deliver Cu into the brain cells. McKenzie et al. (1998) have shown that Cu restores infectivity of scrapie prion (PrPSc), increasing protease resistance after the scrapie prion had been denatured by guanidine.

A number of compounds, including tetrapyrroles (e.g., porphyrin), polyanionic sulfated glycans (e.g., dextran sulfate, pentosan sulfate), and Congo Red, have been shown to interfere with the development of scrapie in mice (Ladogana et al., 1992) and inhibit the formation of protease-resistant PrP in cells (Caughey et al, 1994). There were several studies done in the 1970's that showed a scrapie-like disease to be generated in laboratory animals by feeding them cuprizone, a Cu-selective chelating agent. Treatment of mice with triethylene tetramine dihydrochloride, a Cu-chelating compound, produces neural abnormalities their offspring, including "spongiform changes in white matter" and "reduced myelin development" (Tanaka et al., 1993). This suggests that the removal of Cu from neural cell PrP by soluble chelators could lead to the same pathological symptoms in the brain as caused by TSE disease. In effect, treatment of animals with cuprizone would induce severe Cu-deficiency and the concomitant neuronal degeneration.

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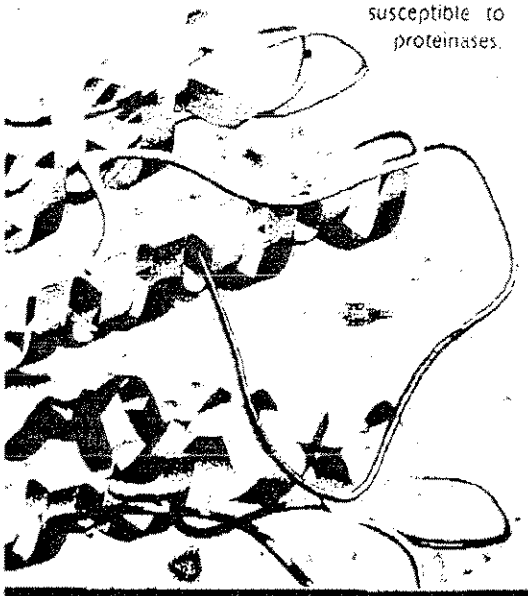
A new theory on BSE and mineral bioavailability

by Philip Lobo

There is a new theory about bovine spongiform encephalopathy (BSE). The theory deals with the role of copper and manganese in the development of prion diseases like BSE and its human variant, Creutzfeldt Jakob disease (vCJD). Larry Berger, PhD, at the University of Illinois recently discussed this theory in a publication from the Salt Institute.


Lobo

The theory suggests that copper plays a role in BSE development. In 1999 Brown showed that copper is part of a normal prion protein, used at the nerve synapse or incorporated in copper-zinc superoxide dismutase. The prion will bind up to four atoms of copper and assumes a structure that is susceptible to proteinases.



However, in 2000 Brown and colleagues showed that the prion was also capable of binding manganese and nickel. When manganese replaced copper, the isoform's most distinguishing characteristic is proteinase resistance and the resulting fibril formation. The isoform's three-dimensional structure changed so that the resulting prion was over 100 times more resistant to proteinase digestion. And, as the manganese bound prion aged, it became increasingly resistant to the proteinase *in vitro*.

If the same thing happens in the animal, the proteinase resistant isoform will accumulate in the brain causing neurological degeneration. This seeding theory helps explain why the disease develops very slowly and then causes the affected animal to deteriorate rapidly, as more isoforms develop and collect in the brain.

The reason for prions

Prions are glycoproteins produced by nerve tissues. "In 1998 Brown and Besinger indicated that the prion may protect the nerves citing its superoxide dismutase activity," explains Berger. "Deactivating harmful oxygen free radicals appears to be an essential role." The prion protein in its normal form is constantly being synthesized by the body and then degraded by proteinase enzymes.

Prion diseases are distinguished by the change in the prion protein from the normal cellular form to an altered isoform. "It appears that when the isoform is introduced into the central nervous system," explains Berger, "it can catalyze the conversion of the normal prion protein to the abnormal isoform."

Minerals' role

It is possible that environmental factors predispose certain animals or humans to prion related diseases. In 2000, Purdy looked into finding a factor common among localized areas associated with scrapie. These areas were Iceland—for scrapie in sheep—Slovakia—for CJD in humans—and Colorado—for chronic wasting disease in deer. The results showed that the soil in these areas is low in copper and high in manganese. Dairy cows fed diets high in soluble protein and sulfur may also have reduced copper absorption. "This reality may help explain why the incidence of BSE has been much higher in dairy cattle than in beef cattle," according to Berger.

Purdey contends that the incidence of BSE is not associated with the feeding of meat and bone meal as much as it is with the use of an organophosphate that inhibits copper absorption.

This does not explain why BSE has become such a problem in the United Kingdom (UK). But, a farmer in the UK, Mark Purdey, studied this problem extensively and made some interesting observations in a British Broadcasting Company (BBC) story. Purdey contends that the incidence of

BSE is not associated with feeding meat and bone meal as much as it is with the use of an organophosphate called Phosmet.

According to Purdey, the Ministry of Agriculture started to force cattle farmers to use the product to control warble fly maggots. One of the traits of Phosmet, according to Purdey, is that it binds copper. If Phosmet bound the cow's copper and the manganese containing prion was present in the meat and bone meal—and absorbed—this may have been the seed prion that initiated the development of BSE. New research by Professor Michel Bounias of the University of Avignon, which has yet to be peer reviewed, suggests that there is a direct link between the spread of BSE in France and the use of a pesticide to kill warble flies.

The relationship of copper and manganese in the development of prion diseases is gaining credibility, according to Berger. Researchers at Cambridge and in France have looked at the copper and manganese concentrations of CJD victims. They found that manganese was 10 times higher in the brains of CJD victims than in the brains of unaffected people.

As a result, in BSE Inquiry, Statement No 638, Brown challenged the idea that people developing vCJD resulted exclusively from eating contaminated meat. He proposes that the imbalance of dietary cations—especially copper and manganese—must be present for vCJD to develop, similarly to cattle. Feeding contaminated meat and bone meal may have magnified the disease, but he believes other nutritional and environmental factors had to be present at the same time to make BSE explode in the UK.

Preventing BSE in the USA

BSE has not been detected in the USA despite active surveillance and serious precautionary measures. Most experts consider the risk of an outbreak in the USA to be very remote. So far, the full story on BSE is not known. And this theory has yet to be proven. Because BSE has an incubation period as long as 10 years, it may take a long time to prove or disprove a theory. However, the role of copper and manganese in the development of prion diseases is gaining credibility. Bergor indicates that the cornerstone of future BSE prevention may be maintaining the proper ratio of copper to manganese in the diet and environment. **FM**

HEAVY METAL FERTILIZE

Cadmium levels dig up debate over phosphate, other fertilizer sou

BY GREG D. HORSTMEIER

Fertilizer, one of the most taken-for-granted inputs in agriculture, has become the subject of hot debate. As part of FARM JOURNAL's ongoing look into fertilizers and the issues that surround them, we've uncovered concerns from agronomists and veterinarians regarding the cadmium content of some sources of phosphate fertilizers, zinc and sewage sludges applied to farmland.

Cadmium is a natural metal—similar to lead, nickel and zinc—found at varying levels in the soil. In that natural state, cadmium is rarely acutely toxic to animals, say published reports on the subject. Yet Tom Swerczek, veterinary pathologist with the University of Kentucky at Lexington thinks cadmium is the common thread in illnesses he's seen in cattle, horses, ostriches and other animals.

"I think a number of immune problems, mineral deficiencies and other undiagnosed illnesses may be directly or indirectly related to cadmium in the environment, and to the problems it can cause in animals," Swerczek says.

If fed high amounts of cadmium, most animals, including humans, will have some form of intestinal upset from the stomach irritation the metal causes before any serious immediate health damage is done, says Robert Smith, a veterinarian and animal nutritionist at Pennsylvania State University. Smith's doctoral dissertation in the mid-1980s examined the influence of cadmium uptake on cattle. Long-term, low levels of cadmium from forages and grains can build up in an animal's body.

As cadmium enters an animal, it binds to proteins and accumulates in tissues such as the kidney and liver. "At higher than normal dietary levels, the body will absorb cadmium instead of similar, but essential, metals such as copper," Smith says. Copper may be in the diet, yet the animals show all the signs of a copper deficiency.

"We've increasingly had troubles with cattle wasting away, getting sick easily, showing all the classic signs of a copper deficiency," says Swerczek. Tests showed the livers and kidneys those animals had cadmium lev from 1 to 2 parts per million (ppr with kidney levels as high as 9 ppm

Swerczek also reports a high in dence of brain lesions in cattle and triches that contained what considers to be high cadmium level liver and kidney.

"It has taken me years of researcl try to figure out what's going on her Swerczek says. "Everything ke pointing to cadmium, and to anim eating forages and grains grown soils with rising cadmium levels."

Where that cadmuim comes fr and how much is too much, is the s ject of great debate among agro mists, chemists and the fertili industry. Swerczek's investigati show one of the culprits in toba country is the practice of sprea surplus tobacco leaves back on tures, or directly feeding excess to co plant parts to livestock.

Tobacco and leaf lettuce are tw the most efficient plants at pulling